MMN IN EARS WITH SENSORI-NEURAL HEARING LOSS

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A Master's Dissertation submitted as part fulfillment for the II year M.Sc. (Speech and Hearing) to University of Mysore

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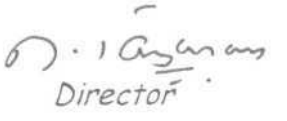
Certificate

This is to certify that the dissertation entitled "MMN in ears with sensori-neural hearing loss" is a bonafide work done in part fulfillment for the degree of Master of Science (Speech and Hearing) of the student with Register No.M 9814.



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This is to certify that the dissertation entitled "MMN in ears with sensori-neural hearing loss" has been prepared under my supervision and guidance.

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Declaration

I hereby declare that this dissertation entitled "MMN in ears with sensori-neural hearing loss" is the result of my own study under the guidance of Mrs. C.S.Vanaja, Lecturer in Audiology, Department of Audiology, All India Institute of Speech and Hearing, Mysore, and has not been submitted earlier in any other University for any other Diploma or Degree.

Mysore *Register No. M 9814*

May 2000

To

The lord Venkateshwara

&

My Family

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INTRODUCTION

Psychophysical experiments involving intensity discrimination as the task, have shown that, in normal hearing conditions, at low sensation levels, the intensity discrimination was poor when compared to that at high sensation levels (Florentine et al., 1993). This issue has been studied extensively, and several hypotheses have been proposed to explain this phenomenon.

However, the physiological data of auditory nerve fibres show that the (auditory nerve exhibits uniform ability to code intensity changes over a wide range of intensities (Delgutte, 1987; as cited in Plack and Carlyon, 1995). These findings have lead to a hypothesis that there are central limitations, which are intensity dependent i.e., they limit the intensity coding differently at various intensities over the dynamic rangeCarlyon and Moore, 1984).

Carlyon and Moore (1984) presented signals with degraded cues at the stimulus level, which are usually given by the auditory nerve to its higher centres for intensity discrimination. Poor performance was observed at moderate intensity levels than at low and high intensity presentations. They hypothesized that in the intensity discrimination of these degraded stimuli, only the higher auditory centres proximal to the auditory nerve, play a major role. Durlach and Braida (1969) also have found similar results, which indicate that the central auditory system is less effective in utilizing the cues available from the auditory nerve, in intensity processing.

Cochlear hearing loss is well known to affect the frequency selectivity of the auditory system (Moore, 1989). The steep growth of loudness with increase in intensity suggests that the hearing impaired ears might have superior intensity resolution compared to normal ears, since a given change in intensity presumably produces a larger change in loudness in the impaired ears (Plack and Carlyon, 1995). Inspite of a dysfunction at the peripheral level, it is not known, why the intensity discrimination is better than that observed in the normal ears. Single neuron recording studies in a chinchilla, after an exposure to noise, have shown that the maximum amplitude of the evoked response is more after the noise exposure, in higher nuclei such as inferior colliculus of the central auditory system (Salvi et al., 1991). From these results it may implied that (higher level nuclei of the central auditory system participate in the loudness recruitment, after a cochlear lesion, in turn resulting in better DLIsJ

/A non-invasive technique for auditory discrimination, that has major contributions from the higher centres of the auditory system may be helpful in examination of the role of these centres in intensity processing after a cochlear lesion. Mismatch negativity (MMN), an electrophysiological correlate of auditory discrimination may be one such test, since studies indicate that MMN originate from the cortical & sub-cortical structures of the central auditory pathway (Alho, 1995)/

A review of literature shows that the MMN has been used to study the impaired processes of auditory attention, memory etc., in degenerative diseases such as the dementia of Alzheimer's type (Pekkonen et al., 1994) and some neurotic disorders such as the obscessive-compulsive disorder (Giard et al., 1990). It has been used to study the development of speech perception in infants and young childern (Kraus et al., 1993). It had also been used in studying the effectiveness of a cochlear implant (Ponton and Don, 1995). However, it has not been used to study the discrimination processes in ears with cochlear lesions/

The aims of the present study were,

a) To examine the effects of degree of cochlear hearing loss on mismatch negativity, and

b) To observe the effects of frequency of the stimuli on the characteristics of MMN in ears with sensori-neural hearing loss.

REVIEW ON INTENSITY DISCRIMINATION

In this chapter, investigations carried out on intensity discrimination in listeners with normal hearing, cochlear-and retrocochlear pathology are reviewed. It comprises of:

* the definition of intensity discrimination,
* the models of intensity discrimination.
* the effects of type of stimulus on DLL in ears with normal hearing,
* the effects of frequency and intensity of carrier tone on DLI in ears with normal hearing, and hearing loss due to cochlear and retrocochlear lesions,
* the electrophysiological evaluation of intensity discrimination.

(A) Definition: Intensity Discrimination:

Intensity discrimination refers to the ability of the auditory system to detect differences in the intensity of two sounds (Plack and Carlyon, 1995). A large number of studies have attempted to measure the threshold of this aspect of auditory function, which has been termed as the "just noticeable difference for intensity" (JNDI) or the "difference limen for intensity" (DLI).

An important concept related to the intensity discrimination is Weber's law, which states that the value of I/I is a constant (K) regardless of the stimulus level, or

where ' I' is the smallest detectable increment in intensity and T is the intensity of the pedestal or standard stimulus^

For. wideband noise, the smallest detectable change in intensity, I, is approximately proportional to the intensity of the stimulus, I. That is. the Weber fraction, is a constant. Within the range of intensities from about 20 dB above the absolute threshold to about 100 dB above the absolute threshold, a plot of 10 log (I) against 10 log (I) (i,e., both expressed in dB) will give a straight line with a slope of 1. In contrast to the results for wideband noise, for pure tones the Weber fraction decreases slightly at high levels, so that a plot of 10 log (I) against 10 log (I) gives a slope of approximately 0.9. This is referred to as the "near miss" to Weber s law as explained by Mc Gill and Goldberg (1968).)

There are several models that attempted to explain the "near miss" phenomenon and also tried to locate the site of coding of intensity discrimination. These models are reviewed below:

(B) Models of intensity coding:

The study of intensity coding is concerned with determining how the ear tells the brain, how intense a particular sound is, or, more specifically, how the physical intensity of a sound is represented, in terms of the activity (Plack and Carlyon, 1995). Intensity coding is inherently related to intensity discrimination. The ability to hear two sounds of 120dB and 130dB, for example, does not imply that these two

intensities are represented differently in the auditory system; they may produce identical percepts. If a listener can detect a difference between the two sounds, however, that difference must be represented at all stages in the auditory pathway between the cochlea and the decision process. The fidelity of the coding mechanism will determine the smallest difference that can be detected. Intensity discrimination experiments are therefore the primary psychophysical tools for testing models of intensity. According to Plack and Carlyon (1995), a successful model of intensity coding has to take account of,

i. the frequency selectivity of the cochlea, so that intensity is encoded independently for different frequency channels,
ii. the firing rates of an auditory nerve fibre in any given frequency region, that codes the whole intensity of a sound,
iii. the limitations of the central auditory system, that should explain the near miss to Weber's law, and
iv. the effects of short term memory on intensity discrimination.

To date no such holistic model is proposed that can take the characteristics of both the peripheral and central auditory systems, but they are based either of the system's individual characteristics. Plack and Carlyon (1995) have described these models as follows:

I. Models based on peripheral systems:

These models describe intensity discrimination purely by the auditory nerve fibers, based on one or more of the characteristics of individual / a collection of nerve fibers.

1) **Models based on dynamic range of the auditory nerve only:**

The human auditory system can detect differences in the intensity of sounds over a very wide dynamic range; as much as 120 dB in normal hearing listeners. This performance is even more remarkable, however, when we consider the information present in the auditory nerve, as measured in other mammals. The majority of auditory nerve fibers, those with a relatively high spontaneous rate, have low thresholds but relatively small dynamic ranges, with most showing a saturation in their firing rate above an intensity of around 60 dB SPL when stimulated by a tone at their characteristic frequency [Palmer and Evans, 1979, as cited in Plack and Carlyon, 1995]. That is to say, increases in stimulus intensity beyond this point will not result in a change in the firing rate of the majority of auditory nerve fibers.

The fact that Weber's law continues to hold even at high intensities has prompted a number of researchers to examine ways in which intensity may be coded other than by the firing rate of nerve fibers, tuned to the pedestal frequency.

2) Models based on spread of excitation:

Although most fibers tuned to the pedestal frequency will be saturated by an intense pedestal, fibers with characteristic frequencies (CFs) remote from the pedestal frequency will receive less excitation and may not be saturated. It has been suggested by Zwicker (1956) [cited in Plack and Carlyon, 1995] that these "off frequency" fibers are responsible for coding intensity at high levels.

(a) Masking spread of excitation:-

The hypothesis of spread of excitation has been tested in experiments that have used masking to limit the information available from "off-frequency" fibers. High-pass noise, low-pass noise, and notched noise centred on the frequency of the pure tone pedestal, have been added to mask spread of excitation and to force listeners to use nerve fibers with CFs close to the pedestal frequency (Viemeister, 1972: Moore and Raab, 1974). These maskers produced a slight increase in the Weber fraction at high intensities, removing the near miss, but performance overall was relatively unimpaired by limiting spread of excitation. Both low-pass noise and high-pass noise were effective in increasing the Weber fraction at high intensities, and notched noise was more effective still at these high intensities in increasing the Weber fraction (Moore & Raab, 1974), suggesting that the spread of excitation on both sides of the excitation pattern is involved in the near miss.

Zwicker (1956, 1970) [cited in Plack and Carlyon, 1995] described a single-band model of intensity discrimination in which performance is assumed to be

determined by the output of the auditory filter in which the change in excitation is greatest. Because of the steeper growth of excitation on the high frequency side of the excitation pattern, with increase in intensity for pure tone pedestals the optimum filter (the one responsible for intensity discrimination), will generally have a characteristic frequency (CF) above the frequency of the pedestal. Florentine and Buus (1981) proposed a multiband version of this model in which information is optimally combined from all regions of the excitation pattern. Although both models predict qualitatively the near miss and its removal by marking with notched noise, the multiband model gives predictions that are in closer quantitative agreement with the psychophysical data.

The results of the masking experiments have been taken as evidence that, although spread of excitation may aid in intensity discrimination at higher intensities, producing the near miss, the auditory system can code intensities over a large dynamic range on the basis of the information from a small range of "off frequency" fibers.

(b) Role of Suppression and Adaptation :

One way for the auditory system to maintain a large psychophysical dynamic range despite the limited dynamic range of majority of auditory nerve fibers would be to adjust the operating ranges of individual fibers according to the input level, so that the intensity of the incoming stimulus always fell on the steep part of their input-output functions. Suppression is a non-linear process whereby intense excitation at

one region of the basilar membrane reduces the excitation in neighbouring regions. It has a very short time constant [Arthur, Pfeiffer, and Suga, 1991, as cited in Plack and Carlyon, 1995], so that suppression occurs only when the suppressor and the suppressee are presented simultaneously. Auditory nerve recordings have shown that a notched noise has a strong suppressive effect on the response of nerve fibers to a pure tone presented in the center of the notch, effectively shifting the rate intensity functions to higher intensities [Palmer and Evans, 1982; Costalupes et al., 1984, as cited in Plack and Carlyon 1995]. In effect, the excitation produced by the pedestal is reduced by the notched noise, and this may increase the apparent dynamic range of the system, accounting, qualitatively, for the results of the experiments described in the previous subsection that used noise to mask spread of excitation. In this way, it is possible that the compressive process on the basilar membrane that produces suppression acts as a form of automatic gain control for wideband stimuli (Plack and Carlyon, 1995).

Making use of the fact that suppression only occurs between simultaneously presented stimuli, Plack and Viemiester (1993) attempted to mask the spread of excitation when avoiding suppressive effect by using non simultaneous masking. They measured intensity discrimination for a brief pure tone pedestal presented in the silent interval between two bursts of an intense masking complex. The masking complex consists of a notched noise and two pure tones with frequencies either side of the pedestal frequency. This complex, masked the spread of excitation without suppressing the pedestal. The results showed that intensity discrimination was

largely unimpaired at high intensities under these conditions, suggesting that suppression is not necessary for the maintenance of a large dynamic range, in a limited frequency region. Although adaptation is, theoretically, a possible additional mechanism that could extend dynamic range, physiological evidence suggests that it does not shift the rate-intensity functions of high-spontaneous rate fibers to higher intensities (Plack and Carlyon, 1995).

3) **Models based on neural synchrony** :

Carlyon and Moore (1984) suggested that, in some circumstances, intensity might be coded by the pattern of phase locking in auditory nerve fibers. Increasing the intensity of a pure tone in the presence of noise can produce an increase in the synchronisation to the fine structure of the pure tone, away from the fine structure of the noise, even though the overall firing rate of the fibers does not change. In other words, the fiber is saturated [Javel, 1981, as cited in Plack and Carlyon, 1995]. In particular, in the notched noise experiment of Moore and Raab (1974), intensity' differences for high level carrier tones may have been detected by virtue of an increase in the degree of synchrony in the firing of a neuron tuned to the pedestal frequency. Carlyon and Moore (1984) tested this hypothesis by measuring intensity discrimination for 30ms pure tone pedestals presented in notched noise for pedestal frequencies of 0.5, 4, and 6.5 kHz. At 6.5 kHz phase locking to the fine structure of the pedestal would be completely absent. Carlyon and Moore demonstrated that there was a large increase in the Weber fraction at high frequencies, but only at medium intensities; at both high and low intensities

performance was still relatively good. Thus, although neural synchrony may play a role in intensity coding, there is sufficient intensity information from other sources at low and high intensities. In other words, neural synchrony is not solely responsible for the large dynamic range observed in these experiments.

4) Models based on rate-intensity functions :

The models discussed previously have indicated that the dynamic range in a single frequency channel is probably much greater than that observed in the high spontaneous rate (SR) fibers. The dynamic range of hearing must depend, therefore, on the minority of auditory nerve fibers with low SRs. These fibers have higher thresholds than the high-SR fibers and have larger dynamic ranges, many of which extend up to very high intensities. A plausible hypothesis is that the high-SR fibers' are responsible for conveying intensity information at low stimulus intensities, and the low-SR fibers are responsible for coding intensity at high stimulus intensities. Plack and Carlyon (1995) referred to this as the "dual population model" of intensity coding. The main drawback of this model is that it has difficulty explaining why the Weber fraction is roughly constant for a wide range of stimulus intensities. On the basis of the dual population model it might be expected that intensity resolution would be much more acute at low intensities than at high intensities. This is both because of the far greater numbers of high-SR fibers than the low-SR fibers and the fact that the rate-intensity functions of low-SR fibers are shallower than those for high - SR fibers. This means that a change in intensity at high intensities will result

in a smaller change in the firing rate of the low-SR fibers than the same proportional change in intensity at low intensities produces in the firing rate of the high-SR fibers.

Viemiester (1988) described a model of intensity discrimination based on the rate-intensity functions of auditory nerve fibers. The first stage of this model calculates the sensitivity measure, d', for a given change in intensity based on the rate-intensity function and the variability in firing rate of a single auditory nerve fiber. The shallower the rate-intensity function and the larger the variability, the smaller the value of d', and the poorer the sensitivity. The performance of a group of nerve fibers is then calculated from an optimal combination of the information from the individual fibers. Viemeister (1988) had calculated the data from physiological data of Liberman (1978) and Evans and Palmer (1980). These data do not obey Weber's law and performance at low-to-medium intensities is predicted to be far superior to that at high intensities. Thus the neural data lead to very poor predictions of human performance. However, when the responses of 10 or 50 fibers are averaged, it leads to better than human performance, over a range of almost 100 dB. These results suggest that sufficient information is available in the firing rates of auditory nerve fibers to encode intensity over a wide dynamic range.

5) **Models based on non-simultaneous masking:**

A recent discovery by Zeng and colleagues (Zeng, Turner & Reikin, 1991; Zeng & Turner, 1992) has aroused considerable interest in the effects of non-simultaneous masking on intensity discrimination. They measured intensity

discrimination for 30 ms pure-tone pedestals presented 100ms after a 90 dB SPL narrow band noise. The Weber fraction was unaffected compared to the value in quiet for low and high pedestal intensities, but was increased by 5-10 dB at pedestal intensities between about 40 and 70 dB. Zeng et.al., argued that the "mid level elevation" might be related to the physiological finding that low-SR fibers take several hundred milliseconds to recover their sensitivity after intense stimulation. During this period, the thresholds of the low-SR fibers will be elevated. On the basis of the dual population model, Zeng et al, suggested that intense stimulation creates a discontinuity in the coding of intensity between the saturation level of the high-SR fibers and the elevated thresholds of the low-SR fibers. This might account for the mid-level elevation under forward masking.

The hypothesis of Zeng et al, appeared very appealing and of significant theoretical interest because, if true, then their experiment would provide the first direct psychophysical evidence for the dual population model. Unfortunately, subsequent experiments have cast doubt on these claims. Plack and Viemeister (1992) demonstrated that an even larger mid-level elevation was observed in backward masking conditions, where the masker cannot have affected representation of the pedestal at the level of the auditory nerve. Furthermore, they showed that the elevation observed in both forward and backward masking could be reduced or removed entirely, by presenting notched noise with the pedestal. They argued that there is no known physiological mechanism at the level of the auditory nerve that can account for the effect of the notched noise and hence that the processes

responsible for the elevation, and its reduction by the notched noise, are located more centrally. Two theories being considered are that

i. forward and backward maskers disrupt the memory trace for the pedestal, or that
ii. the effect is related to variability in the loudness enhancement produced by the masker.

The richness of the cues of representation of intensity in the auditory nerve, and the failure of these models to account for Weber's law, implies that some process central to the auditory nerve must not make optimal use of the neural information (Carlyon and Moore, 1984). Presumably, this 'central limitation' determines discrimination performance in most circumstances and prevents human performance from being better at low and medium intensities than at high intensities, as shown form the electrical recordings of individual auditory nerve fiber activity. These limitations and some other properties of mechanism for intensity discrimination are described in the following section.

IL Models based on central mechansims:-

Plack and Carlyon (1995) have speculated much of the following discussion on central mechanisms involved in the intensity discrimination. First of all, these 'central limitations' were modelled as a constant internal "noise", or variability, added to the decision process and this noise/variability is thought to be independent for each frequency channel (Carlyon and Moore, 1984). This assumption is reasonable if the central noise arises from synaptic transmission throughout the

auditory pathway. While it is naive to assume that there is a single unitary central limitation, it is also naive to assume that there is just one for each frequency channel.

Experiments that have reduced or degraded the information in the physical stimulus available to the auditory system have provided valuable cues as to the nature of central limitations in intensity coding. Carlyon and Moore (1984) went to extreme lengths to remove potential cues for intensity discrimination. They used short tone-burst pedestals presented in notched noise (to mask spread of excitation) at high frequencies (to remove phase locking information) and with the onset and the offset of the tone burst masked with bursts of noise to prevent listeners using "transient" cues, such as the physiological onset response. The result of these manipulations was not an overall degradation in performance at all pedestal intensities, but an increase in the Weber fraction at medium intensities only. They offered two broad explanations for this finding:

i) In the absence of information from spread of excitation and neural synchrony, the coding in the auditor}' nerve is less accurate at medium intensities. Under normal circumstances this is not evident because the central limitations determines. Performance. When the information in the stimulus is degraded sufficiently, however, the information in the auditory nerve becomes the limiting factor and hence the coding deficiency at medium intensities is evident in the psychophysical data. A possible reason whey there might be such a

deficiency is that the basilar membrane, while being relatively linear at low and high intensities, is compressive in the range from 40 to 80 dB. In this region, therefore, the same proportional change in intensity will not produce as large a change in basilar membrane displacement as at low or high intensities. The main problem with the hypothesis is that none of the physiological models demonstrate a coding deficiency at medium intensities although all of the single channel versions show a deficiency at medium intensities. It might be expected, therefore, that degrading peripheral information would degrade performance more at high intensities than at medium intensities.

ii) Some aspect of the central limitation itself is intensity dependent. Reducing the peripheral information might have the effect of this dependency. This issue is further explained in the next section.

Most intensity discrimination experiments employ a two-alternative task in which the listener is required to choose which of two observation intervals separated by an interstimulus interval (ISI), contains the more intense stimulus. These three intervals constitute a trial. This task could be performed in two different ways. First, , the listener could directly compare the intensities of the stimuli in the two observation intervals. This requires that the listener store a representation of the intensity of the first stimulus in short term memory. Second, if the pedestal, or standard, has the same intensity across a number of trials, then the listener may form a long-term representation of this intensity that can be used to perform the task on a

within-interval basis, avoiding a direct comparison of the stimuli in the two observation intervals.

The absence of a substantial effect of ISI in intensity discrimination tasks employing a fixed standard supports the idea that listeners use long-term memory. A short-terra store would be expected to decay over time, producing a large effect of ISI. As an extreme example, Pollack (1955) [cited in Plack and Carlyon, 1995] used an ISI of 24 hours and found only a small deterioration in intensity discrimination performance. The long-term memory cue can be removed by randomly varying the intensity of the standard between trials, so that listeners are forced to make a comparison between the two stimuli within each trial. When this is done, performance consistently worsens with increasing ISI. For a 100 ms, 1 kHz sinusoid, the Weber fraction increases from about -2 dB to 5 dB as the ISI increased from 250 ms to 8 sec. Presumably, this reflects the decay of a short-term memory store. If this is the case, then this memory limitation may be an important component of the central limitation in some circumstances.

Durlach and Braida (1969) described a model of intensity coding that includes two different modes of memory operation, the trace mode and the context-coding mode. In the trace mode, the direct sensations produced by the stimuli are stored. These sensations have the tendency to decay over time, leading to an increase in "memory noise" and accounting for the effects of ISI. In the context coding mode, on

the other hand, intensity is coded relative to a reference intensity; for example, the absolute threshold or the discomfort threshold.

The accuracy of the coding is supposedly dependent on the "distance" on the sensation axis between the sensation of the target stimulus and the sensation of the reference. If manipulations in the signal such as masking, decreasing the signal duration etc., are to degrade the memory trace for the pedestal in some way, then discrimination at low and high intensities may be affected less because the intensity of the pedestal can be context coded with respect to the absolute or discomfort thresholds. This creates a more robust memory trace that is less susceptible to degradation. Thus the context coding hypothesis may provide a mechanism whereby the central limitation may be intensity dependent. In normal circumstances the memory trace is rich enough to be relatively immune to the effects such degradation, however, when the signal is manipulated then the system relies more and more on context coding, which is not effective at medium intensities. Consistent with the relationship of relative intensity to object identification, the context code is often regarded as a "categorical" type of memory trace, so that, for example, in the profile analysis, the listener may categorise each stimulus as "bumped" or "flat" and use these distinctions as the basis for discrimination.

All these models are summarised in table Re. 1.

Table Re.l. Summary of models and hypothesis on processing of intensity changes, in **the auditory system**

Author(s)	Model	Mechanism	Features	Drawbacks
1. Palmer & Evans (1979)	Auditory Nerve Fibers	Change in rate	Increase in Intensity results in increase in the rate of firing	Can explain the intensity discrimination only till 60-70dB, because most fibers are saturated beyond these levels.
2. Zwicker (1970)	A N Fibers	Spread of excitation	At high intensities, fibers whose CFs are away from the frequency of the pedestal detect the changes (off-frequency fibers).	Explains discrimination only at high intensities,
3. Zwicker (1969)	"	Single band model spread of excitation	The off-frequency fibers lying on the high frequency side of the stimulus, detect intensity changes, These fibers, lie in a single critical band.	The data does not closely match the psychophysical data.
4. Florentine & Buus(1981)		Multiband vision of spread of excitation	The oil-frequency fibers lie in several critical bands The output of this model is in close agreement with the psychophysical data.	—
5. Palmer & Evans (1982)	ANFibers	Suppression due to simultaneous masking	A fast non-linear process, because of which the rate of firing, increases in the presence of a noise/tone of different frequency.	It explains intensity discrimination when only two stimuli are present. It cannot explain intensity discrimination at high intensities.
6. Carlyon & Moore (1984)	"	Neural synchrony	It is based on the neural recordings that with increase in intensities, the phase locking increases, even in the presence of noise.	It cannot explain the better DLIs at low & high intensities for frequencies above 6.5 kHz, (Where there arc no phase locking cues), than those at mid intensities

Contd...

Table Re.1 contd....

Author(s)	Model	Mechanism	Features	Drawbacks
7. Plack & Carlyon (1995)	A N Fibers	Dual population model based on the spontaneous firing rates (SR)	The low-level sounds are coded by fibers with high SRs, & the high-level sounds are processed by fibers with low SRs	It can not explain as to why the DLI is constant over a wide range of intensities,
8.Viemeister (1988)	—"—	—"—	The model calculates the sensitivity (d') of change in intensity, & variability in the firing. The data from individual fibers, when considered, poorly reflects the psycho-physical performance while that is averaged from 10-50 fibers, closely agrees with the 'real ear' data Hence, sufficient information is available in the firing rates.	
9. Carlyon & Moore (1984)	Central auditory system	Central noise which is intensity dependent & varies with the frequency of the stimulus	When only the central auditory system is involved (i.e. When the cues for auditory nerve are degraded), the DLI at high & low intensities to better when compared to that at mid intensities. This model is close to the decision making process, hence, its strength.	The exact mechanism for worse performance at moderate levels is not explained,
10. Durlach & Braida (1969)	—"—	Traccmode& context-coding mode of memory operations.	It explains the effects of inter stimulus interval (ISI) on intensity discrimination. The low-level & high-level processing are easy, since they have a reference to the extremes of dynamic range. Hence, can clearly explain, as to why the performance is worse at mid intensity levels (i.e., because there is context for mid levels).	

Knowledge of these models has been used in explaining the facts observed in psychophysical experiments of DLI in ears with normal hearing, cochlear hearing loss or retrocochlear hearing loss. In turn, these studies helped in refining the models. These studies are reviewed in the following section.

(C) Factors affecting DLI:-

As seen in the models, the DLI characteristics vary with the nature and parameters of the stimuli used. Research over this topic is reviewed below.

1. Effects of type of stimulus:-

Zwicker (1975) noted that, most of the intensity discrimination experiments have used one of two techniques termed 'modulation detection' and 'increment detection'. In modulation detection, listeners are required to detect the presence of slow amplitude modulation (AM), threshold being taken as the smallest detectable depth of AM. In increment detection, listeners are required to detect a change in the intensity of a standard stimulus (the pedestal). The pedestal can be presented either continuously or gated with the increment. In the case of a gated increment, the task is usually to discriminate a stimulus containing the increment from one with the pedestal alone, (e.g., which of two stimuli sounds louder).

Zwicker (1975) hypothesized that the 2 different methods are expected to lead to somewhat different values for DL as they involve different stages of the hearing

mechanism. This hypothesis of Zwicker (1975), can be explained by the schematic excitation patterns plotted in the figure 1.

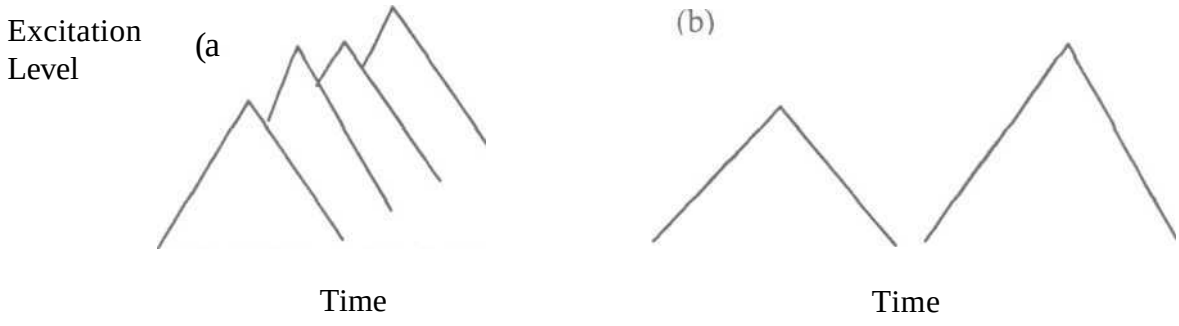

Fig. 1. Schematic excitation patterns, (a) Modulated pure tone.

(b) Pulsed pure tone.

Modulated tones produce the excitation patterns shown in the figure 1. (a), while pulsed tones lead to the excitation patterns deputed in figure l.(b). The difference in the detection mechanisms becomes obvious from the figure 1: with modulated tones, a detector can directly sense an increase or decrease in excitation level. With pulsed tones, however, the detector must first of all determine the excitation level of the first tone and store the resulting value. Then the excitation level of the second tone must be determined and compared to the stored excitation value of the first tone. The task of detecting differences in excitation level is thus much more complicated for pulsed tones than for modulated tones. Presumably, the detection of level differences between pulsed tones involves higher centers of the auditory pathway than modulation detection (Zwicker, 1975).

Dimmick and Olson (1941) and Harris (1963) [cited in Jesteadt et al., 1977] found that procedures based on detection of modulation in an ongoing signal yield different results than those based on discrimination of discrete signals. Bilger et al, (1971) [cited in Jesteadt et al., 1977] obtained level DLs for three listeners as a function of carrier level for four signal ensembles. Carrier level was defined either by a continuous tone or by a 500 ms pulsed tone 1000 Hz. An adaptive sequential procedure, incorporating 4 interval-forced choice method (4 IFC), was used in estimating thresholds. DLs for the continuous carrier tended to be smaller than those for pulsed carrier.

2. Effects of intensity and frequency of pedestal on level difference Iimen (DLI)

The difference Iimen for intensity depends on the level of the carrier tone, however, this can not be generalised to all kinds of signals used in the level difference Iimen experiments.

Zwicker (1952) [cited in Fasti and Schorn, 1981] extensively studied the dependence of the modulation threshold on important stimulus parameters. The threshold for AM of pure tones was found to decrease considerably with increasing sound pressure level of the carrier tone. Moreover, a clear dependence of the modulation threshold on frequency was shown with the AM tones.

In contrast to this behaviour, the discrimination of level differences of pulsed tones was found to be independent of test tone frequency. Moreover, for pulsed

tones, just noticeable level differences decrease only slightly with increasing sound pressure level. To describe this small decrease, Jesteadt et al., (1977) proposed the equation

$$L = (1.644 - 0.0141\ SL / dB)\ dB,$$

Where L denotes the level difference limen and SL the sensation level of the pulsed tone.

Fasti and Schorn (1981) evaluated the level difference limen in listeners with normal hearing and hearing loss groups, using pulsed tones at 250 Hz 500 Hz 1 kHz, 2 kHz, 4 kHz and 8 kHz at 30 dBSL (re: pure tone threshold at that frequency) for 97 observes with normal hearing, the mean difference limen (DL) was 1.5 dB at all frequencies tested, and the interquartile ranges were only + 0.5 dB. In 25 patients with conductive hearing loss, the DL scores were very nearer to those of normal hearing. In subjects with ototoxic hearing impairment, sudden deafness and noise induced hearing loss, the DLs were higher and completely frequency independent. In presbycusis subjects, the DLs were again higher but some persons showed normal DLs. However, in cases with Meniere's disease, the DLs were well above the normal range and in retrocochlear impairment-subjects, the DLs were greater than 4 dB.

Florentine (1983) found that normal hearing subjects show less deviation from Weber's law at 14 kHz than at 1 kHz, when the DLs were measured at 0-, 20-. 40-, 60-, 80-, and 100- dBSL. This study indicated obvious effect of frequency on DL and in turn Weber's law. In the second experiment, intensity discrimination was

measured for a 1 kHz tone at 90- dB SPL in the presence of high pass masking noise whose lower cut off frequencies varied from 6 kHz to 19 kHz. The results indicated that, as the cut-off frequency was increasing from 7 kHz to 19 kHz, the DL at 1 kHz increased by a factor of between 1.5 and 2.0. This suggested that the audibility of very high frequencies is important for intensity discrimination at 1kHz at high intensities. They further confirmed this finding by the third experiment in which the pure tone thresholds were obtained in 12 listeners. Results showed that the DLs at 80 dB SPL correlated with the ability to hear very high frequencies (i.e., HTLs at high frequency). It was concluded that both the test frequency and the high frequency hearing of the subjects may be important variables in pulsed tone intensity discrimination.

Carlyon and Moore (1984) measured the Weber fractions for 500, 4000 and 6500 Hz tone bursts over the intensity' range of 10-90 dB under conditions of masking and no masking. Spread of excitation was restricted by using band stop noise centered at the signal frequency. They observed that for low - frequency tones the intensity DL remains almost constant over a wide range of levels and durations, and the DL is slightly affected by adding band stop noise centered at the signal frequency. For high frequency tones, intensity discrimination deteriorates at intermediate sound levels. They concluded that the large effect of band pass noise suggests that at high frequencies and short durations subjects use information from neurons with CFs remote from the signal frequency. They proposed that subjects maintain performance across level at low frequencies by using a combination of 'firing rate' and 'phase

locking' cues. One or both of these cues is always available, even in the presence of band stop noise. At high frequencies, phase-locking information is absent and the deterioration in intensity DLs at moderate levels (55-70 dB) may be due to firing rate information being impoverished at these levels.

Florentine et al., (1987) measured DLs for 250, 500, 1000, 2000, 4000, 8000, 10,000, 12,000, 14,000 and 16,000 Hz tone bursts as a function of intensity from 10-95 dB SPL. The stimulus duration was 500 ms and inter-stimulus interval was 250 ms. Results for six normal listeners showed individual differences among listeners, but the general trends seen in the average data clearly are present in the individual data. They observed that DLs at all but the highest frequencies are generally smaller at high levels than at low levels. DLs at 8 and 10 kHz are clearly a non-monotonic function of level showing poorer discrimination at moderate levels than at low or high levels. Intensity discrimination is poorer at the high frequencies than the low and middle frequencies.

Florentine et al., (1993) measured level DL of pulsed tones as a function of level in 13 listeners with SN hearing impairment of primarily cochlear origin, one listener with vestibular schwannoma, and six listeners with normal hearing. For normal hearing listeners, the DL decreased with increasing intensity throughout the dynamic range. They also noticed large inter subject variability. In the retrocochlear ears, the DLs were considerably worse than the normal ears. Listeners with lesions of predominantly cochlear origin showed different trends depending on the

configuration of the loss. At low sensation levels, listeners with flat and mildly sloping audiograms showed the same DLs as that of normal listeners. At high levels, the results were varied: some showed better DLs with increasing level, some showed constant performance with increasing level, and some showed worse resolution with increasing level. Listeners with falling audiograms showed two trends: First, the higher the test frequency, the poorer the DL. Second, although DL decreased somewhat with increasing level, there was generally less improvement with increasing level for the impaired listeners than for the normal listener. The group of listeners with rising audiogram configurations showed DLs which decreased very rapidly with level in the impaired ear. Above 10 or 20 dBSL, the impaired ear performed approximately as well as the normal ear. They concluded that, the intensity discrimination at a given frequency depends on the pure tone threshold at the frequency, configuration of hearing loss and pure tone thresholds at high frequencies.

Schroder et al., (1994) obtained Weber fractions for gated 500 ms tones at 0.3, 0.5 1, 2, and 3 kHz, and at levels of the standard tone ranging from absolute threshold to 97 dB SPL in quiet and high-pass noise backgrounds in five listeners with cochlear hearing loss, and in three normal-hearing listeners. DLs obtained in the quiet background decreased as the level of the standard tone increased, demonstrating the near miss to Weber's law. In the high-pass noise background, the listener's reduced ability to discriminate intensity changes, resulted in a nearly constant function of the level of the standard, conforming more closely to Weber's law. The Weber fractions

obtained in quiet from listeners with flat SN hearing loss fell near the mean normal curve. In listeners with falling audiograms, the DLs at all frequencies were within the regions of normal hearing in quiet, but the Weber fractions elevated by the addition of high-pass noise. They concluded that at a given SL (sensation level), the Weber fraction for frequencies in regions of cochlear loss may be considerably better than normal. At low SLs in the presence of high-pass noise, where excitation spread is limited, the Weber fraction for frequencies in regions of cochlear loss are slightly better than normal. This may reflect the steeper rate versus level functions seen in the auditory nerve for CFs in regions of pathology.

From the above review of DLI experiments in ears with normal hearing and those with SN hearing loss following observations can be made:

a) The DLI at frequencies of cochlear hearing loss may be normal / better, when obtained at low SLs as well as at high SLs.

b) DLI, in cochlear hearing loss also depends on the configuration of hearing loss. Sloping hearing loss causes a worsening of DLI at all frequencies.

c) The degree of hearing loss does not has as much influence as the configuration of hearing loss.

(D) **The electro physiological evaluation of intensity discrimination:**

Intensity discrimination may be evaluated objectively by a negative event-related potential (ERP) in the region of 200 ms after the stimulus presentation. Naatanen et. al., (1978) observed this response in unattended conditions and named it

as mismatch negativity (MMN) because of its nature. MMN has been shown to be present when the deviant stimuli were just discriminable from the standard stimuli but not when the difference was not perceptible (Sams et al., 1985). MMN has been obtained for standard and deviant stimuli in a random series, when they differ in frequency, or intensity, or duration, or spatial location or other spectral characteristics as in speech sounds. Alho (1995) concluded in a comprehensive review of cerebral generators of MMN, that a major contribution of supratemporal cortical activity of right hemisphere to MMN elicited by different kinds stimulus changes, in human and some animal cortices. MMN has been suggested to be generated by a neuronal mismatch between a deviant sensory input and a sensory -memory trace representing a preceding repetitive sound. Therefore, the frontal lobe structures also participate in the MMN generation process.

Thus it is clear that, MMN can be used to evaluate the role of central auditory system in intensity discrimination in humans. Keeping the facts observed in the review of DLI experiments, intensity discrimination was evaluated using MMN in the present study.

METHODOLOGY

The aim of the study was to study the mismatch negativity obtained from ears with sensori-neural hearing loss.

(A) SUBJECTS:

Sixteen ears of six male and ten female subjects reported to AIISH, with a complaint of hearing loss, who satisfied the following criteria were chosen for the study.

a) Confirmed sensori-neural hearing loss in the ear to be tested,

b) Pure tone average in the range of 26-55 dB HL,

c) No history of neurological complaints,

d) Should be able to sit quietly at least for half an hour.

Subjects were informed about the procedure and time required for testing. Based on the pure tone average, the subjects were divided in to two groups. Group A included subjects with pure tone average between 26 and 40 dBHL. Group B included subjects with pure tone average between 41 and 55 dBHL. The audiometric and demographic data of these two groups is shown in table R.l.

Table R. 1. The audiometric and demographic data of groups A&B.

Group	Number of subjects	Age range	Mean age	PTA range	Mean PTA
A.	Male Female 4 6	38-61 yrs.	44 yrs	33-40 dBHL	35 dBHL
B.	2 4	45-82 yrs.	41 yrs.	42-51 dBHL	47 dBHL

(B) Instrumentation

The following instruments were used in the study.

* A calibrated GSI-61 two-channel diagnostic audiometer (Grason-Stadler Inc.) with TDH-50p earphones housed in MX-41/AR ear cushions, and a.B-71 bone vibrator was used to diagnose the hearing loss.

* A calibrated GSI-33 middle ear analyzer (Grason-Stadler Inc.) was used in examining the middle ear status.

* The Bio-logic Navigator evoked potential system with EP 317 software (Bio-logic Inc.) was used to generate the stimuli, to record the responses and to analyse MMN. The stimuli were calibrated in nHL.

(C) Test procedure:

Pure tone thresholds were obtained over a frequency range of 250 Hz to 8000 Hz at octave intervals for air conduction stimuli and over 250 Hz to 4000 Hz at octave intervals for bone conduction stimuli.

Immittance evaluation included the measurement of tympanometry and acoustic reflexes to rule out middle ear pathology. Whenever indicated, special tests, electro-physiological and/or behavioural, were carried out to rule out the presence of retrocochlear lesions.

For recording MMN, subjects were seated comfortably in an armed chair and were asked to relax the jaw and neck muscles and also not to pay attention to the stimuli. Since, the test duration lasted for about half an hour, they were asked to indicate at any point of time, if they required a break. Stimuli were presented through TDH-39 earphones, placed in MX-41/AR ear cushions. Four silver chloride electrodes were placed on four sites to pick up the MMN response.

F_{PZ} - Common

P_Z & C_Z - Non-inverting

M_i - Inverting

After cleaning the electrode sites with surgical spirit, and a skin preparing solution, the silver chloride electrodes filled with standard EEG electrode paste were placed and fixed in place with a surgical tape.

It was ensured that the impedance of each electrode was less than 5 k , and the inter-electrode impedance difference was less than 3 k . Each subject underwent four consecutive recordings of MMN for intensity deviance i,e,. at two test frequncies-1000 Hz and 6000 Hz; at two intensity levels of the standard tone-60 dBnHL and 40 dBSL (ref, to pure tone threshold). The stimulus and recording parameters used are described below:

Stimulus	:	Tone burst
Rise time	:	10 ms
Fall time	:	10 ms
Plateau time	:	30 ms
Repetition Rate	:	1.1/sec
Frequency	:	1000 Hz and 6000 Hz
Intensity deviance	:	5dBnHL
Intensity of the standard tone	:	60 dBnHL and 40 dBSL (ref. to pure tone threshold)
Number of Frequent to Infrequent stimuli	:	5:1
Transducer	:	Head phone
Channel 1	:	Cz-Mi
Channel 2	:	Pz-Mi

Time window	:	0ms	to 399.8ms
Filter setting	:		1.1 Hz to 30Hz
Amplifier Gain	:		50,000
Averaging	:		100 infrequent stimuli

(D) Wave form analysis:

- The MMN response was obtained by subtracting the response for the frequent stimulus from the response for the infrequent stimuli, at both Pz and Cz sites.
- For the identification of the MMN true response through visual detection following criterion have been used:

a) MMN is the first negative trough with absolute amplitude less than -0.3|lV in the latency range of N_1P_2 or $P_{2;}N_2$ complex of LLR, and

b) LLR should be present in the unsubtracted frequent and/or infrequent wave form,

c) The negative trough should be followed by a positive peak (absolute amplitude more than +0.3μv).

- The MMN response, thus obtained was analysed for the following parameters:

a) Peak amplitude - The maximum absolute amplitude of the peak of the MMN, with respect to the zero voltage line.

b) Peak latency - The time taken for the peak to occur after the stimulus presentation.

c) Duration - The time lapse between the onset of the negativity till its offset in the following the positive peak.

RESULTS

The data collected from sixteen ears was analysed in terms of wave morphology, peak latency, peak amplitude and duration of MMN. Mean, standard deviation and range was calculated for these parameters for the combined group as well as for groups A & B, separately. To investigate the aims of the study Mann-Witney U test (for unmatched groups) was carried out using NCSS software (version 3.4).

(A) Wave Morphology:

(i) Among the sixteen ears tested, MMN responses could not be identified in all the four recordings of three ears i.e., two ears with mild SN hearing loss and one with moderate SN hearing loss

(ii) In general, the visual resolution of the waveforms was better when the stimulus frequency was 1 kHz rather than 6 kHz.

(B) Descriptive statistics:

(i) The peak latency, peak amplitude and duration, of MMN waveforms obtained from both the groups were combined together to obtain the mean, S.D. and range of each parameter, as seen in tables R. 1., R.2., and R.3.

As shown in table R.I., the mean peak latency obtained for a tone burst of 1000 Hz were 158.63 ms. and 120.25 ms. at 60 dBnHL & 40 dBSL presentation levels of the frequent stimulus. The peak latencies ranged from 106.22 to 221.80 ms. and 66.39 to 179.63 ms. for 60 dBnHL and 40 dBSL stimulation levels, respectively.

For 6000 Hz tone bursts at 60 dBnHL, the mean peak latency was 166.16 ms. in a range from 122.62 to 203.84 ms. The mean peak latency was 127.39 ms. and the range was 65.60 to 214.78 ms. at 40 dBSL levels.

Table R.1: The mean, range and S.D. values of peak latency in ms. for the combined group.

Frequency	1000 Hz		6000 Hz	
Intensity	60 dBnHL	40 dBSL	60 dBnHL	40 dBSL
Mean	158.63	120.25	166.16	127.39
Range	106.22-221.80	66.39-179.63	122.62-203.84	65.60-214.78
S.D.	34.60	34.19	34.61	41.71

Referring to table R.2, the peak amplitude ranged from -5.03 to -0.54 μV and -7.42 to -0.90 μV for 1 kHz tone bursts at 60 dBnHL and 40 dBSL levels, respectively. The mean peak amplitudes were -3.13μV and -3.16 MV at 60 dBnHL and 40 dBSL for 1 kHz tone bursts. At 6 kHz, the mean peak amplitudes were -2.13 μV at 60 dBnHL and -2.17 μV at 40 dBSL. These

values ranged from -5.18 to 0.55μV and -5.08 to -0.85μ V at 60 dBnHL and 40 dBSL, respectively.

Table R.2: The mean, range and S.D. values of peak amplitude (in μV) for the combined groups.

Frequency	1000 Hz		6000 Hz	
Intensity	60 dBnHL	40 dBSL	60 dBnHL	40 dBSL
Mean	-3.13	-3.16	-2.13	-2.17
Range	-5.03 to-0.54	-7.14 to-0.90	-5.18 to-0.55	-5.08 to-0.85
S.D.	1.34	2.05	1.49	1.52

As enumerated in table R.3., the duration values obtained from combined group are shown in table R.3. The mean duration for 1 kHz tone bursts was 68.33 ms. and 78.22 ms. at 60 dBnHL and 40 dBSL levels. The mean duration at 60 dBnHL was 75.25 ms. and at 40 dBSL was 73.47 ms. for tone bursts of 6KHz

Table R.3: The mean, range and S.D. values of MMN duration (in ms.) of MMN for the combined group.

Frequency	1000 Hz		6000 Hz	
Intensity	60 dBnHL	40 dBSL	60 dBnHL	40 dBSL
Mean	68.33	78.22	75.25	73.47
Range	35.15-104.56	39.06-128.87	44.92-121.05	35.93-103.09
S.D.	22.92	26.21	28.35	23.78

To investigate the aims of the study, the parameters of MMN were also analyzed separately for groups A & B.

(ii) The peak latency, peak amplitude and MMN duration obtained from group 'A" (ears with mild SN hearing loss), were as follows:

Referring to table R.4., the mean peak latency at 1 was 148.78 ms. and 122.18 ms. for intensity levels of 60 dBnHL and 40 dBSL (re: pure tone threshold), respectively. The mean of peak latency value was 150.99 ms. and 119.80 ms. at 60 dBnHL & 40 dBSL, intensity levels of 6 tone bursts.

Table R.4: The mean, range and S.D. values of peak latency values (in ms.) obtained from group 'A'.

Frequency	1000 Hz		6000 Hz	
Intensity	60 dBnHL	40 dBSL	60 dBnHL	40 dBSL
Mean	148.78	122.18	150.99	119.80
Range	106.22-199.94	77.32-170.26	122.62-197.59	96.06-150.73
S.D.	31.53	29.48	40.67	21.56

As seen in table R.5., the mean peak amplitudes were -2.87μv and -0.94 μV at 60 dBnHL for 1 and 6KHz tone bursts, respectively. The mean peak amplitudes were -3.67 μ V and -1.97MV at 40 dBSL at 1 & 6 kfrequencies.

Table R.5: The mean, range and S.D. values of peak amplitudes (in obtained from ears with mild SN hearing loss (group A).

Frequency	1000 Hz		6000 Hz	
Intensity	60dBnHL	40dBSL	60dBnHL	40dBSL
Mean	-2.87	-3.67	-0.94	-1.97
Range	-5.03 to -0.54	-7.42 to-1.28	1.22 to-0.55	-4.29 to-0.89
S.D.	1.55	2.30	0.35	1.41

From table R.6., the mean duration values were 69.62 ms. and 78.69 at 1 for 60 dBnHL & 40 dBSL respectively. The duration means were 77.77 ms. & 80.10 ms. at 60 dBnHL & 40 dBSL of 6 tone bursts, respectively.

Table R.6: The mean, range and S.D. values of MMN duration (in ms.) obtained from group 'A'

Frequency	1000 Hz		6000 Hz	
Intensity	60 dBnHL	40 dBSL	60 dBnHL	40 dBSL
Mean	69.62	78.69	77.77	80.10
Range	35.15-104.56	50.02-116.36	44.92-114.20	48.82-100.77
S.D.	25.19	23.81	34.78	20.52

(iii) The characteristics of MMN obtained from group 'B' (ears with moderate SN loss) were are as follows:

Table R.7. demonstrates the statistical measures obtained for peak latency from group B. The mean peak latency was 178.33 ms. & 117.15 ms. at

60 dBnHL & 40 dBSL for 1 kHz tone bursts. The 6 kHz tone bursts elicited the mean peak latencies, 175.25 ms. & 136.87 ms. at 60 dBnHL & 40 dBSL levels.

Table R.7: The mean, range and S.D. of peak latency values (in ms.) obtained from group 'B'

Frequency	1000 Hz		6000 Hz	
Intensity	60 dBnHL	40 dBSL	60 dBnHL	40 dBSL
Mean	178.33	117.15	175.25	136.87
Range	153.0-221.8	66.39-179.63	126.52-203.84	65.60-214.78
S.D.	37.81	44.35	31.53	61.67

The peak amplitude values obtained from group B are summarised in table R.8. The mean peak values were-3.64μV and-2.35 at 2 recordings of 1, kHz. The mean values were -2.8 μ.V & -2.43 μV at 2 recordings of 6 kHz.

Table R.8: The statistical measures of peak amplitude (in μV) obtained from group B.

Frequency	1000 Hz		6000 Hz	
Intensity	60 dBnHL	40 dBSL	60 dBnHL	40 dBSL
Mean	-3.64	-2.35	-2.80	-2.43
Range	-4.21 to-2.81	-4.40 to-0.90	-5.18 to-1.22	-5.08 to-0.91
S.D.	0.74	1.42	1.52	1.83

The MMN duration data from group B was analyzed and found that the mean duration was 65.73 ms. & 69.82 ms. for 60 dBnHL levels at 1 kHz & 6 kHz, respectively. The mean values were 77.48 ms. and 65.26 ms. at 40 dBSL for 1 kHz & 6 kHz tone burst frequencies, respectively.

Table R.9: The statistical measures of MMN duration (in ms.) obtained from group 'B'.

Frequency	1000 Hz		6000 Hz	
Intensity	60 dBnHL	40 dBSL	60 dBnHL	40 dBSL
Mean	65.73	77.48	69.82	65.26
Range	45.68-89.81	39.06-128.87	44.92-121.05	35.93-103.39
S.D.	22.34	32.69	31.06	28.10

(C) Results of Mann-Whitney U test:

To meet the aims, the "Mann-Whitney U test", a non-parametric test for significance of difference, was administered with the help of Number crunching statistical software (NCSS) package, version 3.4.

(i) The first aim was to examine the effects of degree of hearing loss on MMN. The dependent variables were: peak amplitude, peak latency and MMN duration, obtained from all the four recordings, i.e., MMN recorded for two levels at 1 kHz & 6 kHz (for e.g., figures R. 1. to R.4). The independent variable was degree of hearing loss (group A Vs. group B). Comparison of data from groups A & B showed that the mean peak latencies were different. For group B

peak latencies were prolonged for both recordings of 6 kHz and also at 60 dBnHL at 1 kHz. They are not different at 40 dBSL of 1 kHz tone bursts. The mean peak amplitudes of MMN obtained for 6 kHz tone bursts at 60 dBnHL; & was lower for A when compared to that of group B. For other recordings, there is not much of a difference between groups. Results of the "Mann-Whitney U test" revealed that the probability of difference between the means of MMN parameters from groups A & B was less than 95%, for all but 60 dBnHL level. The groups differed significantly ($p<0.05$) in terms of mean peak latencies for 6 kHz tone bursts at 60 dBnHL

(ii) The second aim was to study the effects of frequency on MMN characteristics. The dependent variables were peak latency, peak amplitude and duration of MMN, recorded at both the presentation levels. The independent variable was the frequency of the tone burst (1000 Hz Vs. 6000 Hz). If the frequency effects are observed, in group A, the means of peak amplitude and peak latency were not much different either at 60 dBnHL or at 40 dBSL levels of stimulation. In group B, there was not much a difference for mean and range of peak latency values at either presentation levels. A typical trend in either direction was not observed for latency and duration of MMN. The mean peak amplitudes were different at either presentation levels, and were reduced for 6 kHz tone bursts at

both levels of stimulation. The Mann-Whitney U test was administered separately for the data obtained from groups A & B. The MMN obtained from an ear belonging to group A at different frequencies are shown in figures R.5., and R.6. The analysis revealed no significant difference between the means of MMN characteristics, even at 0.05 level, in either of the groups.

These observations in context with the relevant studies are discussed in the following section.

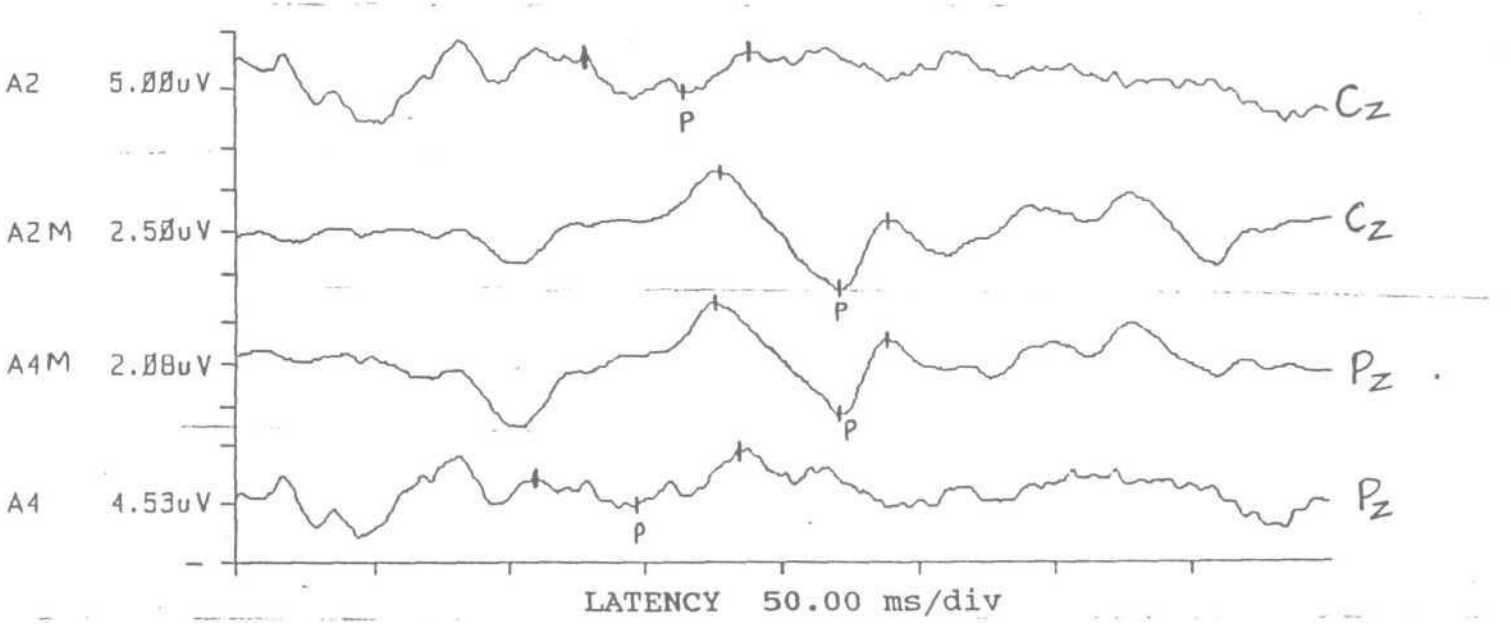

Fig.R.l. MMN waveforms obtained from two ears with mild- & moderate-SN hearing loss for tone bursts of 1 KHz at 60 dBnHL.
A2 & A4: Ear with mild SN hearing loss at Cz & Pz.
A2M & A4M: Ear with moderate SN hearing loss at Cz & Pz.

E → Peak = P

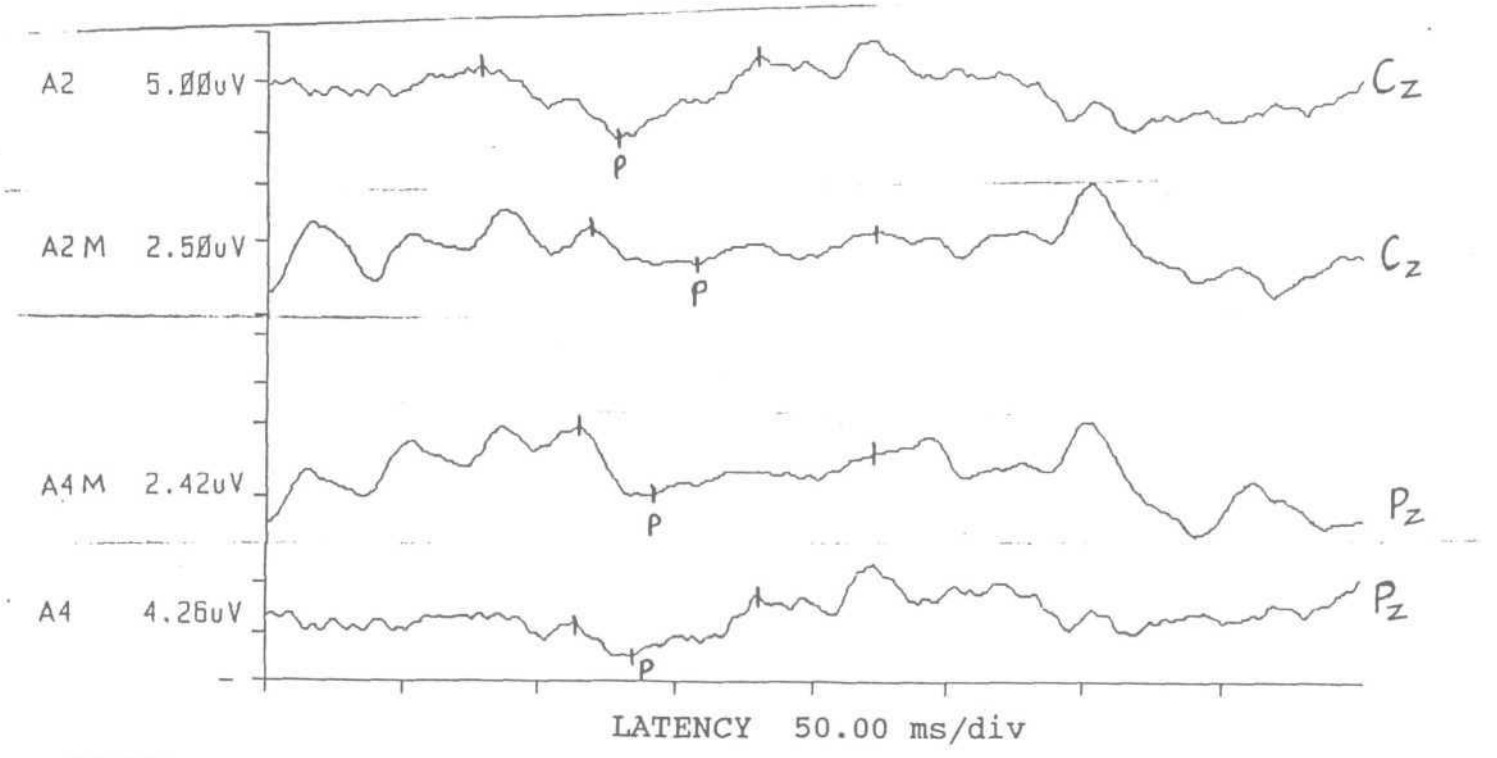

Fig.R.2. MMN responses obtained from two ears with mild- & moderate-SN bearing loss for tone bursts of 1 KHz at 40 dB SL. (re: Pure tone threshold)
A2 & A4: Ear with mild SN hearing loss at Cz & Pz.
A2M & A4M: Ear with moderate SN hearing loss at Cz & Pz.

P → Peak = P

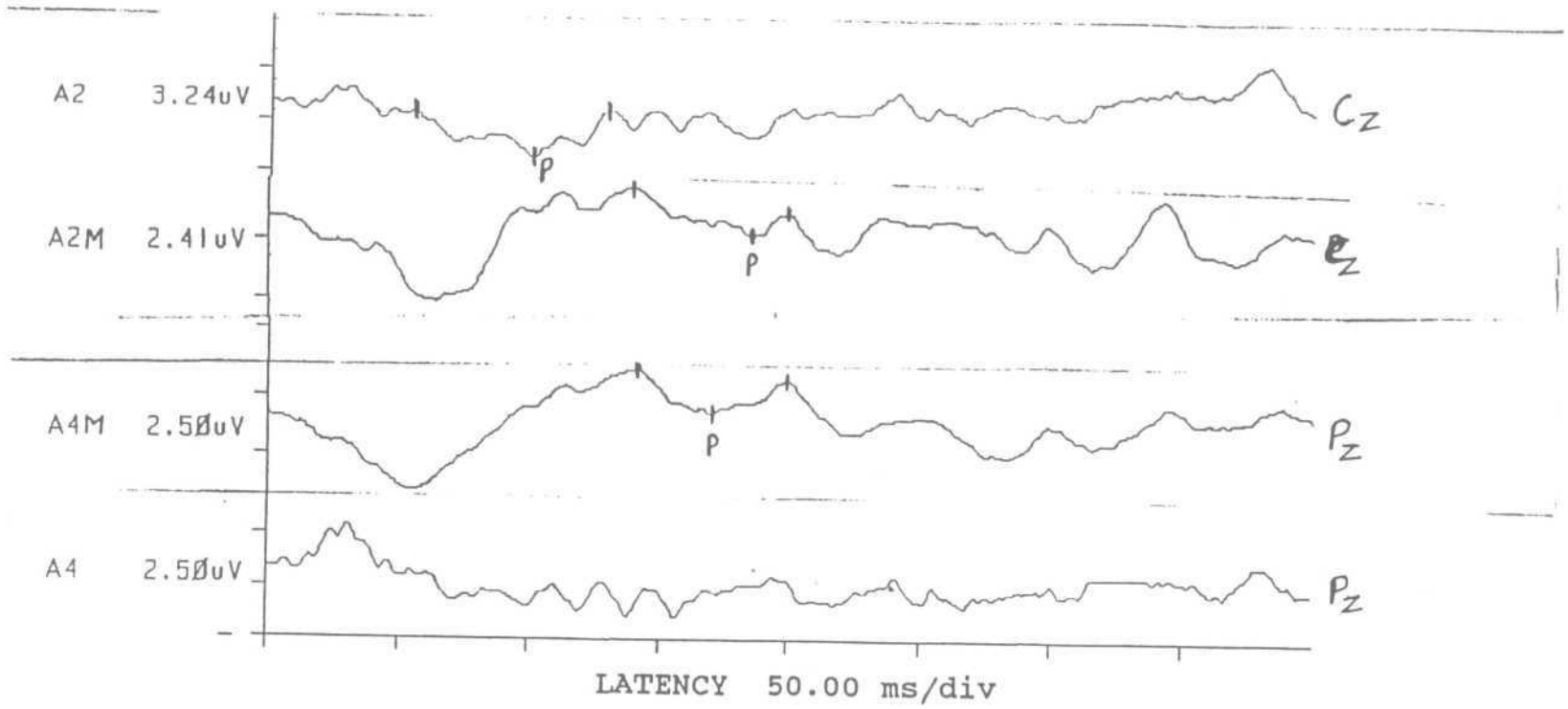

Fig.R.3. MMN wave obtained from two. ears with mild- & moderate-SN hearing loss for tone bursts of 6 KHz at 40 dBSL (re: Pure tone threshold)
A2 & A4: Ear with mild SN hearing loss at Cz & Pz.
A2M & A4M: Ear with moderate SN hearing loss at Cz & Pz.

P → Peak = P

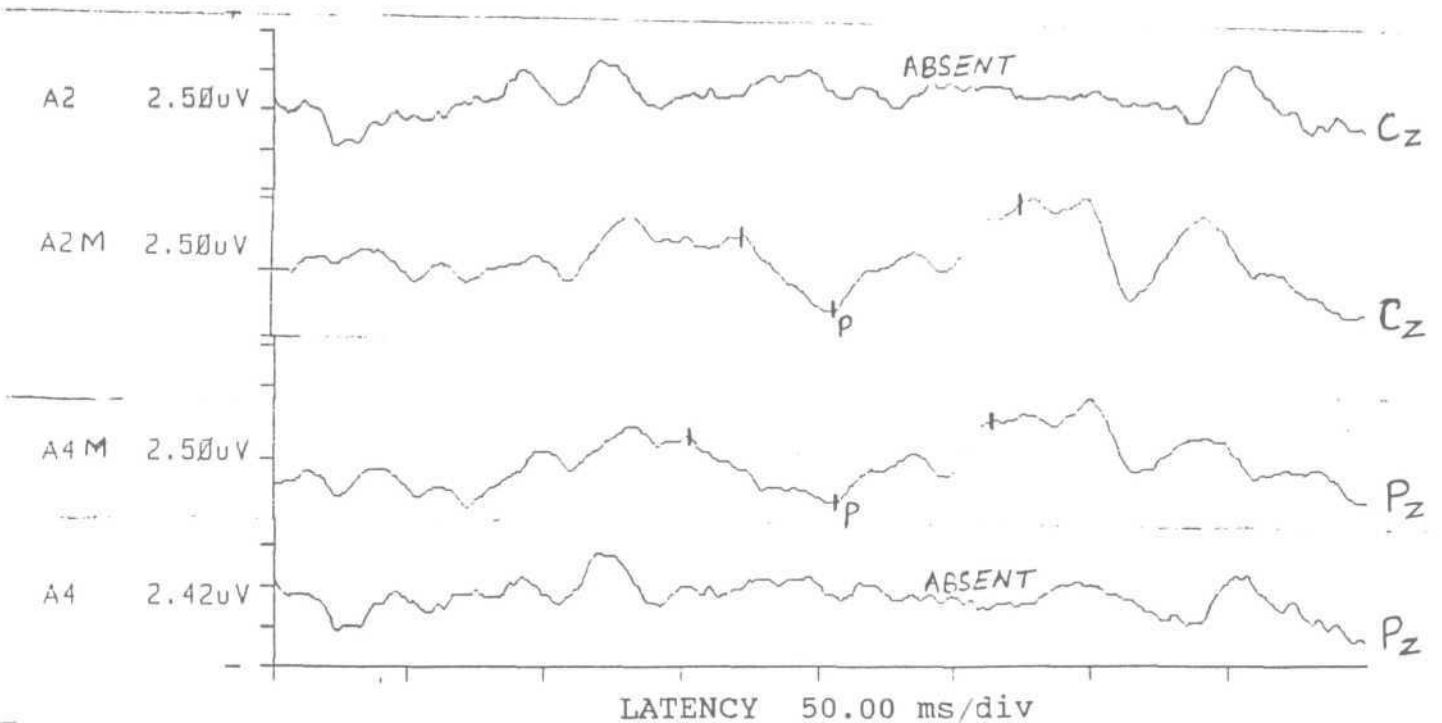

Fig.R.4. MMN obtained from two cars with mild- & moderate-SN bearing loss for tone bursts of 6 KHz at 60 dBnHL.
A2 & A4: Ear with mild SN hearing loss at Cz & Pz.
A2M & A4M: Ear with moderate SN hearing loss at Cz & *Vz.*

E → Peak = P

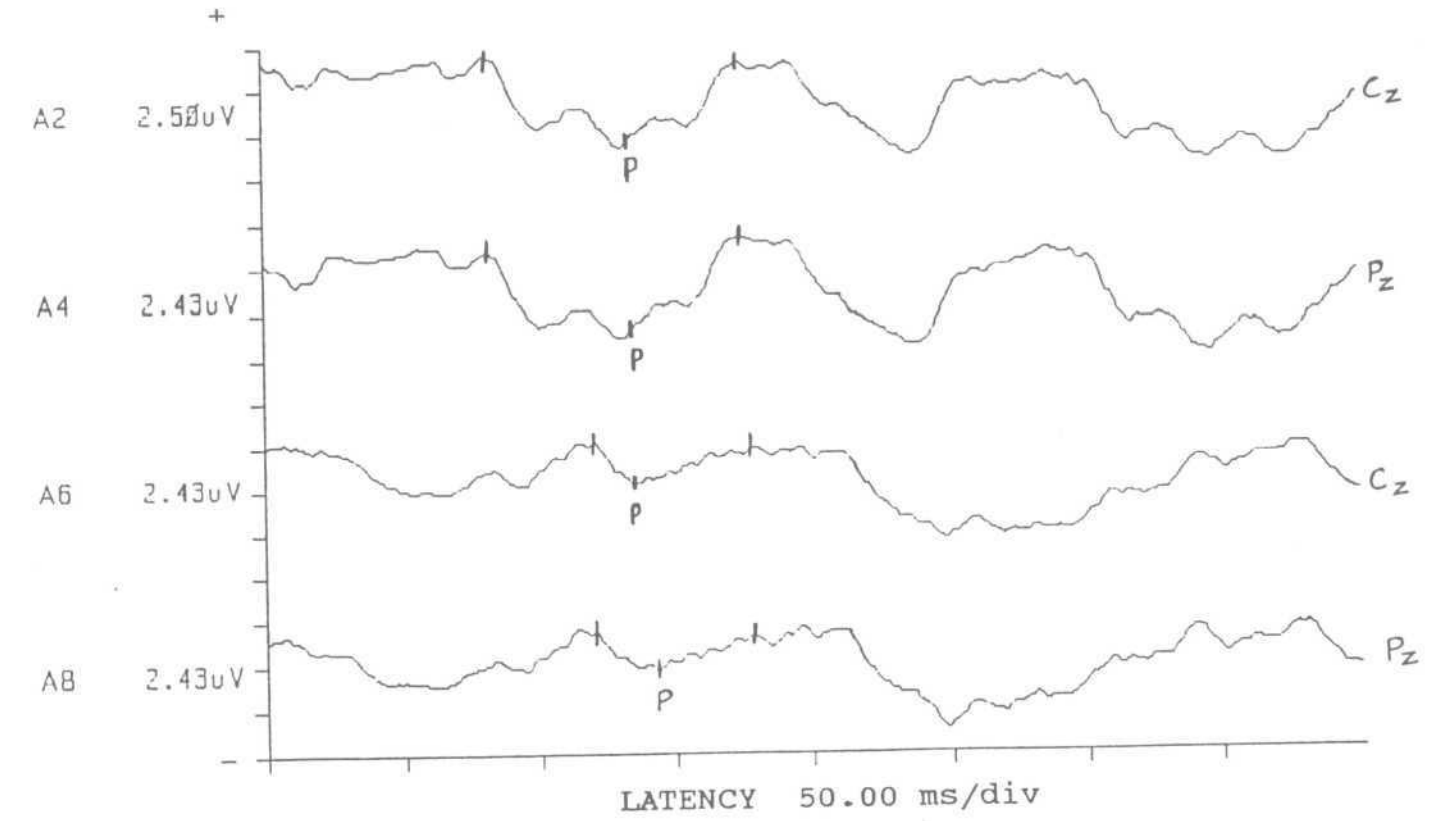

Fig.R.5. MMN obtained from an ear with mild hearing loss.
A2 & A4: For 1 K Hz tone bursts at 60 dBnHL, from Cz & Pz
A6 & A8: For 6 K Hz tone bursts at 60 dBnHL, from Cz & Pz

P → Peak=P

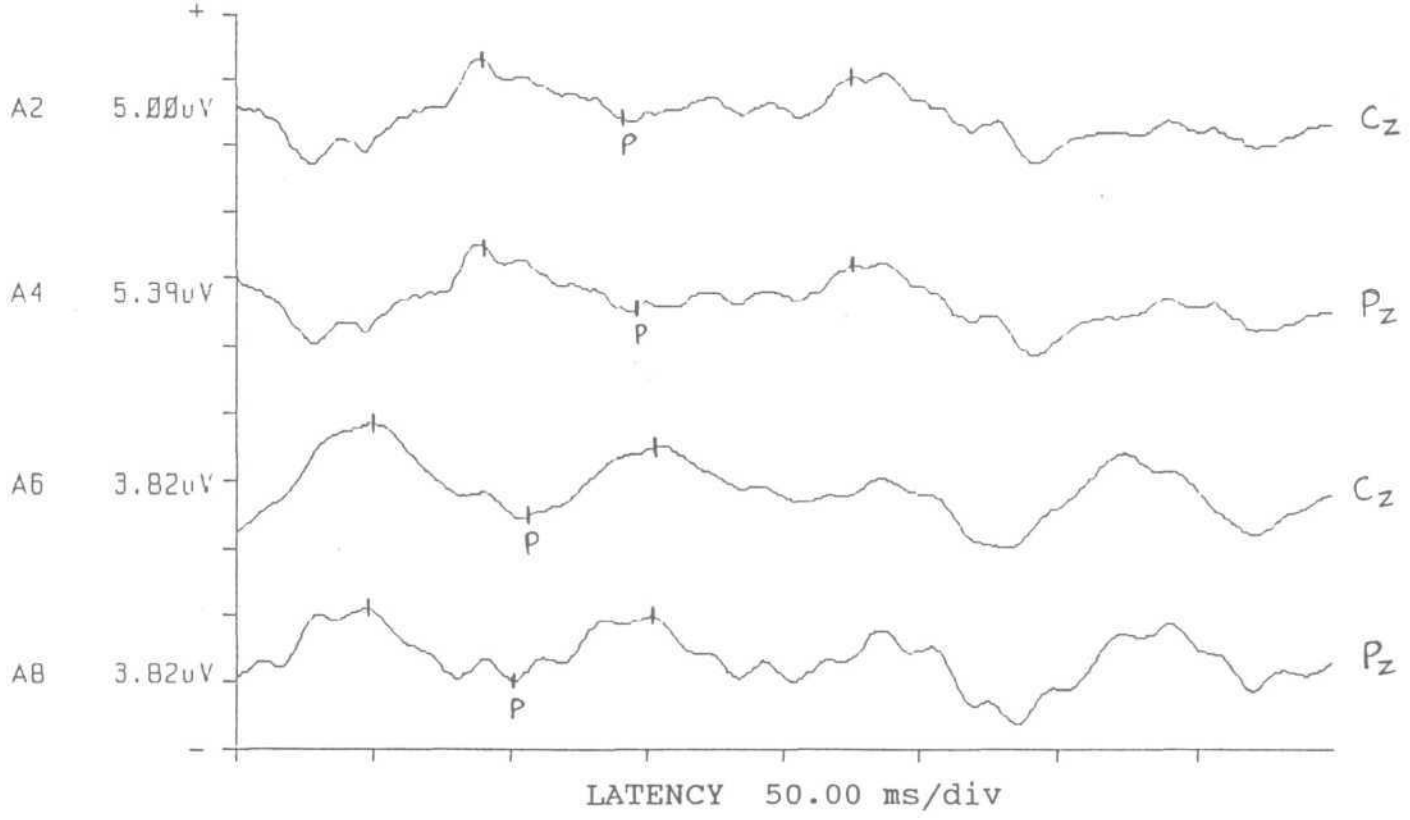

Fig.R.6. MMN obtained from an ear with mild hearing loss.
A2 & A4: For 1 K Hz tone bursts at 40 dBnHL from Cz & Pz
A6 & A8: For 6 K Hz tone bursts at 40 dBnHL, from Cz & Pz

P → Peak = P

DISCUSSION

The results observed in the present study are discussed below in the context of psychophysical and electrophysiological forms of intensity discrimination, studied in ears with normal hearing and cochlear hearing loss.

(i) The three ears, for whom the MMN for intensity deviance, was not observed, had fair speech discrimination scores (>85%), hence the absence of MMN may be attributed to two factors:

(a) the protocol of MMN used, might not have been sensitive enough to detect the MMN (for e.g., the number of averages may be insufficient)

or

(b) the patients might have intrinsic problems in the intensity discrimination which are not reflected upon the speech discrimination score.

However, the percentage of absence of MMN in subjects with SN hearing loss cannot be generalized from the present study, since less number of subjects were included in the study.

(ii) As seen in figure R.5, the visual resolutions for 6 kHz tone bursts was not as good as that for 6 kHz tone bursts. This is supported by other

observations such as. the peak amplitude was more for 1 kHz wave forms rather than that for 6 kHz tone bursts.

The poor visual resolution at 6 kHz may be because of absence of synchronous firing of auditory nerve fibers, for frequencies higher than 5 kHz (Carlyon and Moore, 1984). The better visual resolution with increasing intensities was also reported in normal hearing subjects by Sivaprasad et al., (2000). This may be explained by the hypothesis offered for the generation of MMN itself, which states that MMN amplitude is intensity dependent (Schroger, 1996).

(iii) If the characteristics of MMN obtained in the present study (in ears with SN hearing loss) are compared with those obtained from ears with normal hearing (for 1 kHz tone bursts at 40 dB SL) in Sivaprasad et. al., (2000) study, it is interesting to find that the data do not differ much (shown in Table D. 1).

Table D.1 : Peak latency & peak amplitude of the MMN for 1 kHz tone bursts at 40 dBSL in ears with normal hearing and SN hearing loss.

	Peak latency (in ms.)		Peak amplitude (in μV)	
	Mean	Range	Mean	Range
Present study	120.25	66.39-179.63	-3.16	-7.42 to -0.90
Sivaprasad Et al., (2000)	168.93	56.33-180.12	-2.42	-6.92 to-1.34

From this comparison, it may be inferred that the MMN is least affected by the presence of a cochlear hearing loss. Mc Pherson (1996) also observed that late latency responses (LLRs) are not affected by the cochlear hearing loss.

(iv) Statistical analysis also showed that MMN parameters were not significantly affected by the degree of hearing loss except at 60 dBnHL for 6 kHz. The reason for the presence of a significant difference between peak latencies for 6 kHz tone bursts only at 60 dB levels is not clear. It has been reported in the literature that DLI is more affected by the configurationof hearing loss rather than degree of hearing loss (Schroder, et al., 1994).

(v) Even though the MMN at 6 kHz has prolonged peak latency and less peak amplitude, when compared to that at 1 kHz, the difference was not statistically significant. These results again parallel the trends observed in DLI experiments that DLI remains almost constant (with large individual differences), at any frequency in ears with normal hearing or cochlear hearing loss (Fasti and Schorn, 1981).

Iyengar et al., (2000) also reported similar findings in their study using MMN in listeners with normal hearing. From this, it may be inferred that low frequency tone bursts yield better MMN responses that the high frequency tone bursts.

Overall, it could be concluded from this study that MMN in ears with mild to moderate flat SN hearing loss is not different from that of the normal hearing. Hence, MMN may be used in central auditory evaluation, even in the presence of flat SN hearing loss.

SUMMARY & CONCLUSIONS

Psychophysical studies have shown that both the peripheral (Florentine & Buus, 1981) and central auditory mechanisms (Carlyon & Moore, 1984) are involved in intensity processing. These studies observed that subjects with cochlear hearing loss perform equally well in discriminating intensity changes at low - and high - sensation levels (Florentine et al., 1993), whereas normal hearing subjects performed better at high sensation levels than at low sensation levels (Florentine, 1983) of pulsed tones. In other words, the peripheral hearing loss does not affect intensity discrimination.

Mismatch negativity (MMN) is an electrophysiological tool for sound discrimination. MMN is a negative potential observed in the latency region of around 200 ms. for a train of pulses in an odd-ball paradigm. It can be elicited for a change in intensity (Naatanen et al., 1989), frequency (Sams et al., 1985), direction of sound (Paavilainen et al., 1989) etc. Near-field electro-physiological recordings in animals and magneto-encephalographic studies in humans (Alho, 1995) have shown that MMN has both cortical and sub-cortical generators.

On these grounds, this study on ears with sensori-neural hearing loss was taken up to examine,

(i) the effects of degree of hearing loss and

(ii) the effects of frequency of tone burst,

on the MMN elicited for intensity deviance.

Sixteen ears with sensori-neural hearing loss (group A: ten ears with mild SN hearing loss, and group B: six ears with moderate SN hearing loss) were included in the study. They were presented tone bursts of 1 kHz & 6 kHz at 60 dBnHL & 40 dBSL (re: pure tone threshold) intensity levels, in four successive recordings, using Bio-logic Evoked Potential System (version 3.4). One hundred responses to deviant stimuli were recorded from which the recordings for frequent stimuli were subtracted to obtain MMN. Rigid criteria were employed to identify and accept a waveform.

Results showed that only thirteen ears exhibited recognizable MMN responses. It was observed that the wave morphology was better for 1 kHz tone bursts than those for 6 kHz tone bursts. The statistical analysis of MMN showed,

(i) no significant difference between groups A & B, in the mean peak latency or peak amplitude for all but one recording, viz., for 6 kHz tone bursts at 60 dBnHL levels,

(ii) no significant difference between responses obtained for 1 kHz & 6 kHz tone bursts, either for mean peak latency or peak amplitude, at either levels.

From this study it may be concluded that-

1) MMN in cochlear hearing loss is not different from that of a normal hearing ear,
2) Similar to DLI, MMN is not affected by the degree of hearing loss,
3) MMN is not affected by the frequency of the tone burst in patients with flat SN hearing loss, and
4) MMN may be used in neuro-diagnosis, even in patients with SN hearing loss.

The study has following limitations:

1) Subjects with mild and moderate flat SN hearing loss only were employed. Hence, the results may not be generalized to other degrees and configurations of hearing loss, and

2) These results may be limited only to MMN elicited for intensity deviance. MMN for other type of deviances are not studied.

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